Mediators of Inflammation

Hindawi

Special Issue on

Mediators of Inflammation in Myeloproliferative Neoplasms: State of the Art

CALL FOR PAPERS

Myeloproliferative neoplasms (MPNs) are heterogeneous group of chronic clonal diseases characterized by the excessive production of mature cells of one or more of the myeloid lineages. Recent advances in the biology of MPNs have greatly facilitated their molecular diagnosis, since almost all MPN patients present with mutation in the *JAK2*, *MPL,* or *CALR* genes. Yet the precise role of these mutations in the pathogenesis of the different subtypes of MPNs is not elucidated. Moreover, complications range from mild clinical symptoms such as itching or weight loss to thrombotic events, fibrosis of the bone marrow, splenomegaly, and acute myeloid leukemia. Some of these complications can be linked to the chronic inflammation associated with MPN disease, and the JAK inhibitor clinical trials showed that the reduction of symptoms linked to inflammation was beneficial to patients. Moreover, there is evidence that, for subsets of MPN patients, inflammation may be independent from or/and precede acquisition of mutations in *JAK2*, *MPL,* or *CALR* genes. Thus, better understanding of the causes and molecular mechanisms that underlie chronic inflammation in MPNs seems necessary to improve the treatments currently proposed to MPN patients. To achieve this aim, we propose a new analysis, focusing on inflammation, of the large body of data accumulated in MPNs over the past ten years. This new approach will bring new insights in the pathophysiology of MPNs and highlight new approaches for the therapy of these diseases.

Therefore we invite investigators to contribute original research articles and review articles that will describe, analyze, or summarize the biological and clinical findings accumulated with regard to inflammation in MPNs, with the aim of reaching consensus on the main cytokines and molecular mechanisms that underlie inflammation in MPNs. Then novel therapeutic strategies that target inflammation in addition to the main MPN mutations will be discussed. Note that, here, the term ”inflammatory” refers to both pro- and anti-inflammatory cytokines, and the chemokines may be included in the cytokine family.

Potential topics include, but are not limited to:

- The important inflammatory cytokines/chemokines deregulated in MPNs, as possible predictive markers of disease severity
- Inflammatory cytokines/chemokines as “fuel” for clonal MPN progenitor cell growth
- Inflammatory cytokines/chemokines as drivers of MPN mutations
- Murine model systems for MPNs–emphasizing the role of inflammation
- Congenital genetic factors influencing inflammation in MPNs in humans
- Epigenetic regulation of inflammatory cytokine/chemokine expression or signaling
- Inflammation and increased risk of thrombosis in MPNs
- Inflammation and myelofibrosis in MPNs
- Inflammatory markers as predictors of complication in MPNs
- Possible nongenetic causes of inflammation in MPNs
- Effects of current drugs or combinations of drugs acting on inflammation in MPNs
- Novel therapeutic strategies targeting inflammation in MPNs

Authors can submit their manuscripts via the Manuscript Tracking System at http://mts.hindawi.com/submit/journals/mi/mpn/.

Lead Guest Editor
Sylvie Hermouet, Université de Nantes, Nantes, France
sylvie.hermouet@univ-nantes.fr

Guest Editors
Hans C. Hasselbalch, University of Copenhagen, Copenhagen, Denmark
hans.hasselbalch@dadlnet.dk

Vladan P. Čokić, University of Belgrade, Belgrade, Serbia
vl@imi.bg.ac.rs

Manuscript Due
Friday, 26 June 2015

First Round of Reviews
Friday, 18 September 2015

Publication Date
Friday, 13 November 2015